operant-like behavior of orienting microorganisms, for example, doesn't seem to be under stimulus control (Staddon, 1983), and it is perfectly possible to design an operant mechanism that is context independent (Staddon & Zhang, 1991). So the question is certainly worth asking.

Finally a comment on the authors' question "Are neural networks capable of simulating the effects of nondifferential as well as differential operant contingencies?" (p. 202). As McCulloch and Pitts (1943/1965) showed many years ago, even very simple neural networks are general computing devices of the same order as the Turing machine, and hence are capable of simulating any well-defined process. The scientific issue, therefore, is not whether a given data set can be simulated by a neural network (it can), but whether a given simulation is the simplest and best—truest—model for that data set. What is "true"? Francis Bacon quotes Jesting Pilate asking that question in another context, but Pilate "stayed not for an answer," perhaps because it is not a question that has (as the mathematicians say) a "closed-form solution."

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BIOLOGICAL SUBSTRATES OF OPERANT CONDITIONING AND THE OPERANT–RESPONDENT DISTINCTION

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At the outset I should identify myself as a fellow advocate of the views of Donahoe and his colleagues—as someone who shares their selectionistic approach to behavior, admires their work, and embraces their positions on many specific issues. In particular, I enthusiastically endorse the main organizing idea: that complex behavior "is best understood as the cumulative product of the action over time of relatively simple biobehavioral processes, *especially selection by reinforcement*" (p. 193, emphasis mine). And with regard to the important issue of the nature and complexity of the reinforced response, Donahoe et al. and I hold the same minority position. Together we reject the common supposition that the "whole" response (or its complex neural substrate) can be identified as the functional unit of reinforcement. Rather, we assume that the unit of reinforcement is some sort of infinitesimal response element or be-

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havioral atom (Skinner, 1953), whose biological substrate is presumably cellular or subcellular in scale.

Nevertheless, our positions differ substantially with respect to the set of questions collectively termed "the S-R issue" in this essay. The central hypothesis of the authors—as aptly restated by Shull (1995)—"is that the fundamental effect of reinforcement is to select an environment–behavior relationship rather than to increase the emission rate of the reinforced response" (p. 353). Accordingly, the behavioral atoms of Donahoe and Palmer (1994) are conceived as elementary stimulus–response units, whose connection weights can be increased by the release of the reinforcement transmitter dopamine into the synapses of *coactive* pre- and postsynaptic elements. Furthermore, the unified reinforcement principle in their formulation applies equally to operant and Pavlovian conditioning. For these reasons, the Donahoe–Palmer hypothesis was characterized by Shull (1995) as "unconventional in some interesting respects" (p. 353). After all, the operant–respondent distinction is commonly thought to be at the heart of Skinner's thinking on these matters, and Skinner (1953) has indicated explicitly that "Operant behavior, in short, is *emitted,* rather than *elicited*" (p. 107). Indeed, in later writings, Skinner (1981) even proposed that the evolution of operant conditioning must itself have required the parallel evolution and availability of "a supply of behavior . . . which has little or no relation to [eliciting or releasing] stimuli" (p. 501).

In response, Donahoe et al. acknowledge that, indeed—after Skinner and others had firmly established control by consequences as an empirical fact—the defining postulate of classical S-R psychology (that eliciting antecedents control all behavior) became untenable. But how can this conclusion be squared with the hypothesis that "what is selected is always an environment–behavior relation, never a response alone" (p. 196)? "The apparent incongruity," the authors argue, "arises from a confusion of levels of analysis. . . . control by consequences (as opposed to antecedents) stands as a behavioral law, but we propose (at another level of analysis) that the effects of those consequences are implemented by changes in synaptic efficacies" (p. 196).

Although Donahoe et al. repeatedly assert "that it is a mistake to categorize accounts at the behavioral level by one's view of the underlying biology" (p. 197), they nevertheless seek to reinterpret apparently contradictory neurophysiological findings from my own laboratory (e.g., Stein, 1994; Stein & Belluzzi, 1988, 1989; Stein, Xue, & Belluzzi, 1993, 1994). My colleagues and I found that the spontaneous bursting rate of individual cells in hippocampal slices was progressively increased in a dose-related manner by locally applied, burst-contingent microinjections of dopamine or other reinforcing transmitters or drugs. General pharmacological stimulation of bursting could be ruled out because the same injections given independently of bursting were ineffective, and because contingent (or noncontingent) injections of glutamate—an excitatory transmitter not associated with behavioral reinforcement—failed to increase and even suppressed hippocampal bursting. At the same time, the glutamate injections sharply increased the frequency of solitary spikes. We have interpreted these findings to mean that dopamine and other chemicals can increase the spontaneous bursting activity of neurons by a novel cellular mechanism (in vitro reinforcement or IVR) that is analogous to the strengthening of emitted behavior by reinforcing consequences. In short, IVR was conceptualized as a cellular analogue of operant conditioning (Stein et al., 1993).

Donahoe et al. offer "an alternative interpretation of these same facts . . . that is consistent with our view that reinforcers affect input–output relations and not output alone" (pp. 196–197). Their reinterpretation is based on the premise that IVR should not be viewed as a novel mechanism, but rather that it arises as a variation or manifestation of long-term potentiation (LTP, a well-established model of synaptic plasticity; see review of Bliss & Collingridge, 1993). According to this alternative explanation, the increased bursting induced by chemical reinforcement in the brain-slice experiments "may reflect a heightened sensitivity of the postsynaptic neuron to the release of the neurotransmitter glutamate by presynaptic neurons" (p. 197). Unfortunately, this ingenious and admirably detailed idea is probably incorrect; as already noted, applications of glutamate over a wide

dose range do not increase hippocampal bursting rates (as the alternative interpretation implies they should), but instead strongly suppress them.

In my opinion, IVR and LTP are not mere variations of a common mechanism of synaptic plasticity—they are separate processes. If so, it is possible that their dissimilar neurophysiological properties, when expressed in behavior, may underlie in part the operant–respondent distinction. Although calcium-dependent signaling mechanisms are involved in both processes, IVR and LTP appear to depend on different types of Ca^{2+} channels. The relevant Ca^{2+} channel (NMDA channel) for LTP in the CA1 area is well established (Bliss & Collingridge, 1993); this receptor-operated Ca^{2+} channel is activated by glutamate in conjunction with membrane depolarization. On the other hand, the L-type Ca^{2+} channel seems to be critical for IVR (Stein et al., 1994); this voltage-gated Ca^{2+} channel is activated by membrane depolarization, but only if the channel protein is phosphorylated.

Evidence that implicates the L-type channel in IVR includes the following: (a) L-type channels control the generation of calcium spikes in hippocampal CA1 and CA3 neurons (Kostyuk, 1989), (b) L-type channels are located in hippocampal CA1 cell bodies and cluster in high density at the base of major dendrites (Westenbroek, Ahlijanian, & Catterall, 1990), (c) influx of Ca^{2+} through hippocampal L-type channels regulates gene transcription through a distinct signaling pathway (Bading, Guity, & Greenberg, 1993), thus providing a possible mechanism for long-term reinforcement effects, and (d) L-type channels must be phosphorylated in order to open when the cell membrane is depolarized (Armstrong, 1989); this property could reasonably provide the hippocampal pyramidal cell with a mechanism for modulating calcium fluxes in response to external (reinforcing) signals. The tentative identification of the L-type channel as a significant protein target of the cellular reinforcement process has suggested a plausible and testable molecular hypothesis of IVR and (by extrapolation) operant conditioning (Stein, 1994).

My molecular model is based on the premise that the phosphorylation of L-type Ca^{2+} channels is the ultimate step in the reinforcement of hippocampal bursting responses.[1] More precisely, I propose that the normally rapid dephosphorylation of L-type channels after nonreinforced bursts is prevented by burst-contingent applications of dopamine or other reinforcing agents. Hippocampal bursts are made up of a few initial sodium spikes followed by a succession of calcium spikes (Jensen, Azouz, & Yaari, 1994; Schwartzkroin & Slawsky, 1977; Wong & Prince, 1978). The latter are mediated by voltage-gated L-type channels, which open in response to membrane depolarization (especially that produced by the Na^{+} spikes) if the Ca^{2+} channel protein is phosphorylated. The enzymatic addition or removal of phosphate esters alters the confirmation and thus changes the activity state of many nerve cell proteins (Nestler & Greengard, 1984).

Molecular Hypothesis of In Vitro Reinforcement

Nonreinforced bursting. Following a burst of nonreinforced calcium spikes, a "protective" intracellular cascade is activated to reduce the probability of further bursting (Figure 1) (Armstrong, 1989). This arrangement is thought to be self-protective, because each burst of calcium spikes introduces Ca^{2+} into the cell, and because high levels of intracellular Ca^{2+} are toxic. The burst-induced rise in intracellular Ca^{2+} activates the calcium-dependent enzyme calcineurin, which rapidly dephosphorylates the recently active Ca^{2+} channels that participated in the burst. Calcineurin can itself dephosphorylate the channel protein, but it acts mainly indirectly via inactivation of a key inhibitor (DARPP-32) of the principal dephosphorylating enzyme (phosphatase-1) of L-type channels. Gluta-

[1] Hippocampal pyramidal cells are not thought to be the sole targets of the in vitro reinforcement process. Following Skinner (1953), we have proposed that operant behavior arises from the collective action of a population of infinitesimal response elements or behavioral atoms that serve as the functional units of reinforcement (Stein & Belluzzi, 1982, 1988; Stein et al., 1994). At the biological level, atoms of behavior are assumed to arise from the bursting activity of specialized "reinforceable" neurons (e.g., hippocampal CA1 cells) that are localized in the target fields of dopamine, opioid peptide, or cannabinoid reinforcement systems. Reinforceable neurons thus have a wide distribution in the brain.

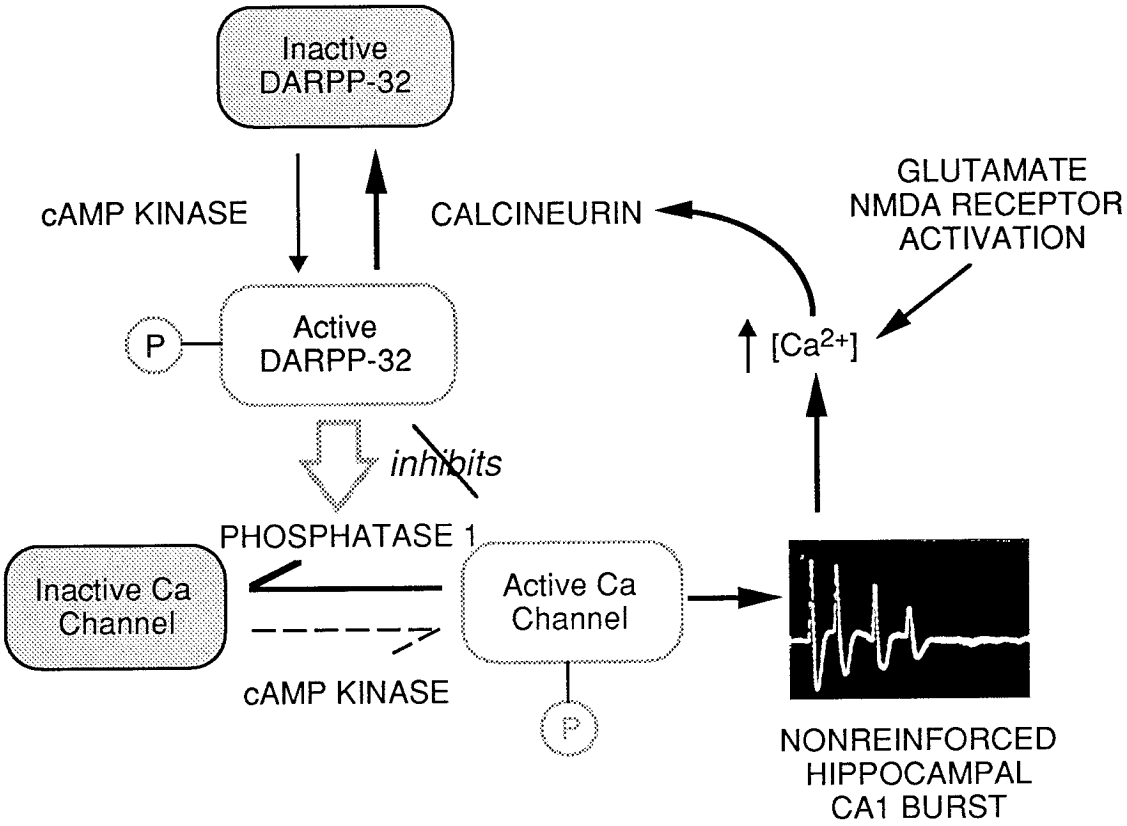

Fig. 1. Inactivation (dephosphorylation) of calcium channels by nonreinforced bursting or glutamate. Elevation of intracellular Ca^{2+} by a nonreinforced burst of calcium spikes or application of glutamate activates the calcium-dependent phosphatase, calcineurin, which in turn dephosphorylates and thus inactivates DARPP-32 (dopamine- and cAMP-regulated phosphoprotein). Such inactivation of DARPP-32 indirectly dephosphorylates the L-type calcium channels by releasing the principal dephosphorylating enzyme (phosphatase-1) of these channels from inhibition. Phosphorylation is denoted by the letter P in a circle.

mate also elevates intracellular Ca^{2+} by stimulation of NMDA receptors. Effective doses of this transmitter would therefore activate the calcineurin pathway, and the resulting dephosphorylation of Ca^{2+} channels should reduce hippocampal bursting rates, as we in fact have found (Stein et al., 1993). The glutamate-induced increase in the frequency of single spikes (i.e., Na^{+} spikes), which we also observed, may be explained by the simultaneous stimulation of non-NMDA (AMPA) receptors that control sodium channels.

Reinforced bursting. According to my hypothesis, if bursting responses are closely followed by stimulation of dopamine D1/D5 receptors, the protective calcineurin pathway will be overridden and dephosphorylation of the recently activated Ca^{2+} channels is thereby prevented (Figure 2).[2] The central feature of any useful hypothesis of IVR must be a plausible molecular explanation of the burst-dependent nature of dopamine's reinforcing action. As in the case of behavioral reinforcers, response-independent applications of dopamine are not reinforcing; hence, in our account, they should not prevent the dephosphorylation of Ca^{2+} channels. What is needed most critically to complete the hypothesis is a molecular coincidence detector, able to respond selectively to the conjunction of burst-

[2] Activation of dopamine D2, D3, and possibly D4 receptors is also reinforcing (Stein et al., 1994), presumably via an alternative pathway for the phosphorylation of calcium channels involving the arachidonic acid cascade (Piomelli et al., 1991), but this aspect of the hypothesis is beyond the scope of the present paper.

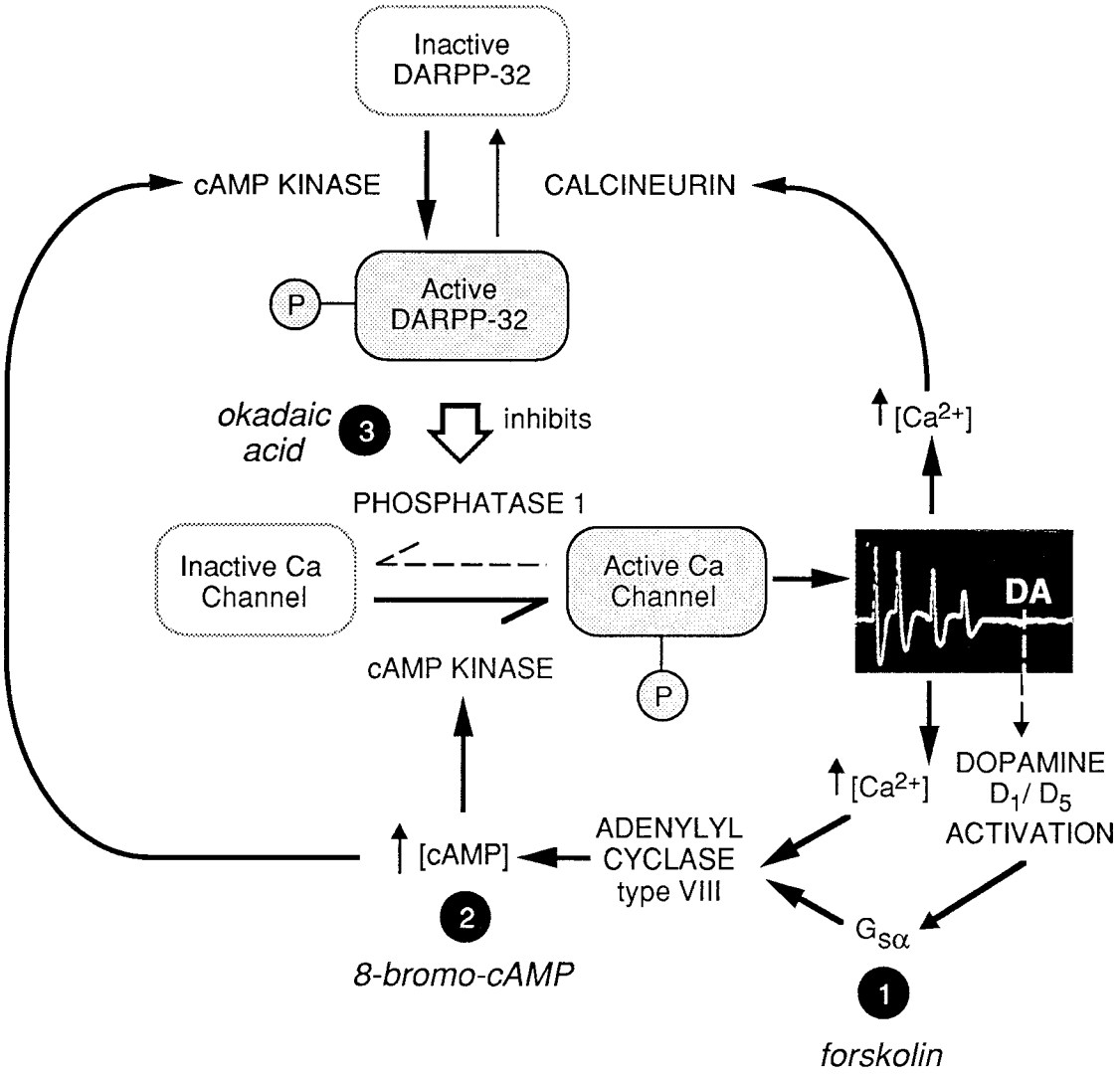

Fig. 2. Molecular hypothesis of in vitro reinforcement: Burst-contingent application of dopamine (DA) overrides the calcineurin cascade shown in Figure 1 and thus prevents the dephosphorylation of recently active L-type calcium channels. The conjunction of burst-induced Ca^{2+} elevation and dopamine D1/D5 receptor stimulation (via the stimulatory G protein subunit, $G_{s\alpha}$) synergistically activates type VIII adenylyl cyclase, causing a sharp rise in cAMP. The resulting activation of cAMP kinase overrides Ca^{2+}-channel dephosphorylation both directly, and also indirectly, by activation of the potent phosphatase-1 inhibitor, DARPP-32. For further explanation, see text.

ing activity and dopamine receptor activation. I propose that the enzyme type VIII adenylyl cyclase performs this function.

Adenylyl cyclase, the enzyme that synthesizes cyclic AMP (cAMP), is the prototypical second messenger generator; indeed, the concept of signaling by second messengers originated with the discovery of the role of cAMP (for reviews, see Nestler & Greengard, 1984, and Cooper, Mons, & Karpen, 1995). The ubiquitous cAMP-dependent protein kinase (cAMP kinase) pathway accounts for the hormonal control of many cellular events; included among these is the phosphorylation of L-type Ca^{2+} channels. Eight types of adenylyl cyclases have been cloned to date, and each has been shown to be regulated by a variety of influences. Surprisingly, one of these adenylyl cyclases—type VIII—exhibits the precise biochemical properties and re-

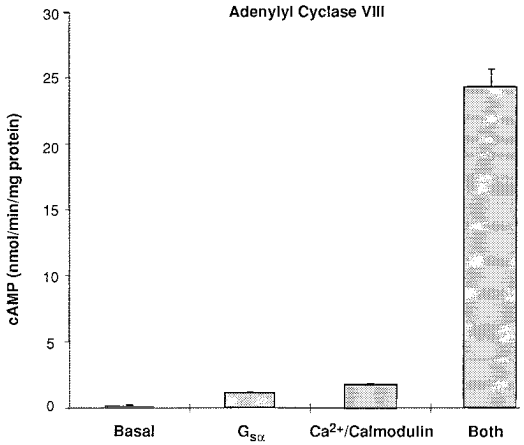

Fig. 3. Synergistic action of Ca^{2+}-calmodulin and the activated α-subunit of stimulatory G protein ($G_{s\alpha}$) on type VIII adenylyl cyclase. Bars show mean cAMP accumulation from embryonal cells expressing rat type VIII adenylyl cyclase after no treatment (basal), addition of each regulatory molecule alone, or combined treatment (both). After Cali et al. (1994).

gional and subcellular localization required for our molecular coincidence detector. The highest concentrations of type VIII immunoreactivity are found postsynaptically in hippocampal CA1 dendritic spines "in intimate association with sites of calcium ion entry into the cell" (Cooper et al., 1995, p. 421); furthermore, type VIII is the only member of the adenylyl cyclase family that responds synergistically to Ca^{2+} and dopamine (via the stimulatory G protein, G_s) (Cali, Zwaagstra, Mons, Cooper, & Krupinski, 1994) (Figure 3). Thus, it can be anticipated that the conjunction of burst-induced Ca^{2+} elevation and dopamine D1/D5 receptor stimulation would readily and selectively activate type VIII adenylate cyclase; the cAMP generated would need to diffuse only a short distance before activating its enzymatic target (cAMP kinase), known to be anchored in high concentrations in the same dendritic spines. Such activation would override the calcineurin cascade and prevent the dephosphorylation of the Ca^{2+} channels in those spines (Figure 2). Finally, the important negative action of burst-independent dopamine is nicely explained by the fact that, in the absence of elevated Ca^{2+}, the response of type VIII adenylyl cyclase to D1/D5 activation will be inadequate.

The model is being tested at various steps of the proposed cascade. Three such IVR tests (each denoted by a black circled number in Figure 2) are in progress. In the first test, microinjections of forskolin (a G_s-mimicking activator of adenylyl cyclase) are substituted for dopamine as reinforcement for CA1 bursting. It is anticipated that forskolin will function as a typical in vitro reinforcer and will produce burst-contingent, but not burst-independent, increases in hippocampal bursting. Similar IVR experiments also are being performed with a membrane-permeant cAMP analogue (8-Br-cAMP, Test 2) and a phosphastase-1 inhibitor (okadaic acid, Test 3). Both 8-Br-cAMP and okadaic acid exert their effects at late stages of the phosphorylation cascade and thus bypass the coincidence-detecting action of type VIII adenylyl cyclase, which occurs at an earlier step. Hence, according to the model, these agents should not require a contemporaneous Ca^{2+} signal to be effective, and both should facilitate bursting whether administered on a burst-contingent or burst-independent schedule. So far, all three tests have yielded promising results.

What are the main implications of this body of work for the S-R issue and the reinforcement hypothesis of Donahoe et al.? If eventually validated and extended to other target regions of the brain's reinforcement systems, the molecular model would provide a detailed explanation of dopamine's reinforcing action at the cellular and subcellular levels. The key feature is the precise specification of a coincidence-detecting molecule (e.g., type VIII adenylate cyclase) that reacts selectively and exclusively "to the contiguity between the bursting of the postsynaptic neuron and the introduction of the neuromodulator" (p. 197). This two-term molecular interpretation of reinforcement thus seems quite consistent with Skinner's views on control by consequences and the operant–respondent (emission–elicitation) distinction, and it may indeed "more transparently parallel behavioral laws than the accounts we have offered" (p. 196).

Having said this, I should like to close with an expression of support for the emphasis Donahoe et al. correctly place on the difficult problem of operant discrimination and the role of the discriminative stimulus. Skinner (1953) himself has pointed out that operant behavior ubiquitously and "almost necessari-

ly'' comes under the control of discriminative stimuli, according to a ''three-term'' contingency (p. 108). Nevertheless, he continues:

> The relation between the discriminative operant and its controlling stimulus is very different from elicitation. Stimulus and response occur in the same order as in the reflex, but this does not warrant the inclusion of both types in a single ''stimulus-response'' formula. The discriminative stimulus does not elicit a response, it simply alters a probability of occurrence. (p. 110)

I am intrigued by the thought that my molecular hypothesis might be extended along the lines followed by Donahoe et al. to reinterpret our IVR experiments, but then used instead to suggest a biological explanation of operant discrimination. The broadened hypothesis focuses on the previously noted facts that (a) the initial spikes in a burst are nonreinforceable Na^+ spikes, and (b) these spikes can be generated by glutaminergic non-NMDA synaptic activation. The initial sodium spikes, of course, are not by themselves able to trigger the calcium spikes that complete the burst; in addition to depolarization of the postsynaptic membrane, a sufficient number of phosphorylated Ca^{2+} channels must be available in the target zone. I propose that the initial sodium spikes in the burst may serve as the biological mediators of discriminative stimuli. Discriminative stimuli gradually assert their powerful control over behavior after many instances of differential reinforcement. The paired presentation of discriminative and reinforcing stimuli leads inevitably to their strong association through Pavlovian conditioning. Accordingly, the extended model must ensure that the sodium spikes are intimately associated with dopamine reinforcement, but the connection should be mediated by a Pavlovian (LTP-like) rather than an operant (IVR-like) mechanism. Donahoe et al. may have identified just such a process based on the LTP research of Frey, Huang, and Kandel (1993). Incorporation of their idea into the model provides it with a plausible Pavlovian-like mechanism by which dopamine D1/D5 activation could act in conjunction with NMDA stimulation to enhance the sodium spiking of the postsynaptic cell to coactive presynaptic (non-NMDA) glutaminergic input. If so, it perhaps would be interesting to employ these mechanisms in a computer simulation of discriminated behavior.

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MELIORATION AND CONTIGUITY

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Donahoe, Palmer, and Burgos make a number of arguments: Molar relations should be understood as the outcome of local processes; reinforcement is not simply the strengthening of responses but also involves the stimuli present at that time; operant and classical conditioning are not distinct, but are separated only on the basis of what kinds of events are reliably present when reinforcement is presented; modeling (in this case by means of a neural network) can be productive in terms of integrating a number of behavioral phenomena.

A number of these issues tie in with an account of melioration (Herrnstein & Vaughan, 1980) in terms of strengthening by contiguity. Consistent with Donahoe et al., I believe that it is possible to derive melioration from the more basic processes advocated by Skinner. In an experiment using concurrently available alternatives, an alternative can gain value by pairing with reinforcement, whether the reinforcement is response produced or not (e.g., using concurrent variable-time [VT] VT schedules and only requiring a changeover response); time spent without reinforcement in the presence of that alternative drives its value toward zero. From these assumptions, one can deduce that the value of an alternative is a strictly monotonically increasing function of rate of reinforcement in its presence. Given two or more such alternatives, changeover responses can then be viewed as increasing or decreasing in strength, depending on whether they make a transition from a lower to a higher, or from a higher to a lower, situation. This strengthening model (presented in Vaughan, 1982), in a nutshell, allows one to deduce the process of melioration, and in turn account for behavior on concurrent variable-interval (VI) VI, concurrent VI variable-ratio (VR), and concurrent VR VR schedules. The fact that a changeover delay is often required to prevent rapid alternation, with a duration similar to the duration of unsignaled delays that will reduce responding to a low level (Williams, 1976), suggests that the strength of changeovers is being maintained by the transitions from one conditioned reinforcer to another, rather than by food presentations on the alternative being changed to.

On the other hand, Donahoe et al.'s argument that operant and classical conditioning are the same processes, distinguished only by what event is reliably contiguous with reinforcement, may require some modification. For example, consider the Rescorla–Wagner model (Rescorla & Wagner, 1972),

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